

REMARKS

Applicant has deleted claims 55-61 in order to overcome the double patenting rejection. Applicant has amended independent claim 37 to specify that the encoded peptide is an N-terminal fragment which has an antioxidant characteristic that is close to full-size Prx V1hum. Support for this limitation can be found on page 6 lines 15-18 of the original specification wherein it is disclosed that the N-terminal fragment of peroxiredoxin Delta Prx V1hum has a similar antioxidant effectiveness as natural Prx V1hum. Applicant has also amended the claims or added claims directed to the specific antioxidant protection provided by the Prx fragment of claim 37. Support for these limitations can be found on pages 10-15 of the original specification.

Claims 42-44 have been amended to overcome the rejection under 35 USC 112, second paragraph. The limitations directed to the antioxidant activity of SEQ ID NO:2 or SEQ ID NO:4 have been deleted. Accordingly, the rejection under 35 USC 112 should be withdrawn.

Reconsideration of the rejection under 35 USC 103 based upon the Kang reference is requested. The N-terminal fragment Delta of Prx V1hum which includes a nucleotide sequence of SEQ ID NO:4 and has a length of 177 amino acids as recited in Claim 37 of the present application has an antioxidant activity which is close to the antioxidant characteristic of full-size Prx V1hum. This is an unexpected property not known prior to the Applicant's invention. The isolation of this Prx fragment and use of the fragment to treat various pathology-inducing exogenous and endogenous factors by introducing the compound into the intercellular space is a novel, non-obvious improvement. The Prx fragment of Claim 37 provides improved antioxidation properties for treatment of pathology-inducing exogenous and endogenous factors including bacterial or viral infection, burn, frostbite, wounds, fractures, concussions or exposure to

radiation, due to a difference in its size and molecular weight as compared to natural peroxiredoxin Prx VIhum. The Prx fragment is more readily permeable into the intercellular space providing improved effectiveness. Furthermore, the compound for treatment comprising a purified recombinant protein or fragment thereof will not induce an immune response since the structures are identical to natural PrxVIhum protein.

Prior to Applicant's invention it would have been expected that the removal of fragments from the complicated tertiary structure of peroxiredoxin VI molecule would lead to substantial, if not complete, loss of the antioxidant activity. The 177 a.a. Prx fragment of claim 37 discovered by Applicant which has antioxidant activity that is close to the full-size Prx VIhum was surprising and unexpected.

The Examiner acknowledges that the cited Kang reference provides no teaching or suggestion to utilize a fragment of human peroxiredoxin. The Examiner states that it would have been obvious to one of ordinary skill in the art to prepare a nucleic acid molecule encoding shorter fragments while preserving regions that encodes Cys 47 and Cys 170 to maintain its peroxidase activity. However, Applicant asserts that a person with ordinary skill in the art would have no motivation to prepare a nucleic acid molecule encoding the Prx fragment of recited claim 37 because the unique properties of this peptide for treatment of pathology-inducing exogenous and endogenous factors was unknown and unexpected at the time of the invention.

The Kang reference discloses peroxiredoxins containing cysteines at positions Cys 47 and Cys 170 which are substituted and compared with the wild type protein. Peroxiredoxins containing cysteines at positions Cys 47 and Cys 170 are characteristic of Prx1-Prx5. In contrast, the present invention is directed to Prx6 which is more effective in treating bacterial or viral infection, burn, frostbite, wounds, fractures, concussions or exposure to radiation because all of these factors involve apoptosis of epithelial or

cutaneous cells where the level of Prx6 is higher than that of Prx1-Prx5. Accordingly, the Kang reference does not make obvious the subject matter of Applicant's invention.

Paigen WO9843666 (Oct. 8, 1998) teaches a peroxiredoxin fragment having a length of 114 amino acids beginning from a.a. 26 to 90. Research has shown that peroxiredoxin fragments having a length of less than 177 amino acids have a considerably lower antioxidant activity than fragments of at least 177 a.a. The fragment taught by Paigen is missing the beginning 25 a.a. portion of Applicant's Prx fragment specified in Claim 37 of Applicant's invention. The Paigen fragment also contains portions (SEQ ID NOS: 6 and 7) which have no antioxidant activity. While Paigen states that peroxiredoxin fragments can be used for increasing the antioxidant activity, this statement was merely hypothetical at the time and not supported experimentally. The fragment described in the Paigen reference will have a low antioxidant activity which is not suitable for the treatment purposes of Applicant's invention. No person with ordinary skill in the art would have expected when viewing the Paigen reference that the 177 a.a. long polypeptide comprising an N-terminal fragment DELTA of Prx VIhum as claimed in Claim 37 of the present application would have an antioxidant activity which is close to full-size Prx VIhum.

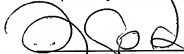
The cited Kang reference as well as the prior art disclosed by Applicant teach use of peroxiredoxins for diagnostic purposes or treatment of diseases such as autoimmune or neurodegenerative diseases, but the prior art does not teach the effectiveness of peroxiredoxin or fragments thereof for treating pathology-inducing exogenous and endogenous factors such as bacterial or viral infection, burn, frostbite, wounds, fractures, concussions or exposure to radiation by introducing peroxiredoxin or fragments thereof in the intercellular space.

A person with ordinary skill in the art would solely have been motivated to search

for peroxiredoxin fragments for diagnosing and determining the amounts of peroxiredoxins in a mammal organism; to develop genetic constructs for improved expression of peroxiredoxins in cells of a mammal, or to prepare a pharmaceutical composition for treating a whole organism or organs thereof. There was no motivation at the time of the invention to obtain a fragment for treating pathology-inducing exogenous and endogenous factors by placing them in the intercellular space.

Accordingly, Applicant's invention, as amended, is non-obvious over the prior art and withdrawal of the rejection under 35 USC 103 is requested.

Respectfully submitted,

A handwritten signature in black ink, appearing to read 'J. Cord', is written over a horizontal line.

Attorney for Applicant
Janet A. Cord (Reg. No. 33,778)

LADAS & PARRY LLP
26 WEST 61ST STREET
NEW YORK, NY 10023
Telephone – (212) 708-1800
Facsimile - (212) 246-8959

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